A STUDY ON ANANLYSIS OF VALIDITY OF LATERAL PLACENTAL LOCATION IN THE PREDICTION OF PRE ECLAMPSIA

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CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON ANALYSIS OF VALIDITY OF LATERAL PLACENTAL LOCATION IN PREDICTION OF PRE ECLAMPSIA"** submitted by Dr.N.Dhivya to the Faculty of Obstetrics and Gynaecology, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree (Obstetrics and Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance

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Date :

CONTENTS

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ACKNOWLEDGEMENT

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INTRODUCTION

INTRODUCTION

Hypertensive disorders represent the most common medical complication of pregnancy. It complicates up to 7-10% of pregnancies of which pre-eclampsia / Eclampsia constitutes 70%, and Chronic hypertension 30%. Pre-eclampsia has been recognized as pathological entity since the time of Hippocrates and ancient Greeks.

Pre-eclampsia complicates approximately 5 - 8% of pregnancies and is a major cause of maternal and perinatal morbidity (SIBAI et al, 1997, 5-8% ACOG 2002).

Pre-eclampsia and Eclampsia contribute 12% of all maternal deaths in the developing countries (WHO 1999).

It is said that pre-eclampsia, eclampsia contributes to death of a woman every 3 minutes worldwide. Infants of women with severe Pregnancy induced hypertension has 5 fold increase in mortality compare to infants of normotensive women. Pre-eclampsia is a multiorgan disorder, and usually recognized by new onset of hypertension and proteinuria appearing in the second half of pregnancy.

Hypertensive disorders complicating pregnancy represent one facet of a complex disease process. Gestational hypertension, pre-eclampsia, eclampsia majority of these conditions are preventable.

This has led to the interest in screening. Screening the deliberate examination of substantial segments of the population in search for disease at its earlier stages, is a logical extension of the role of preventive medicine. If we wish to prevent such disorder we must seek ways of preventing (or) ameliorating the disease process. In preventing this disorder the most important factor is lack of timely prediction.

Preeclampsia occurs only in the presence of placenta . Noninvasive doppler velocimetric studies of the uterine arteries in the second trimester reveal that abnormal wave forms indicating defective uterine perfusion is primarily a consequence of placental implantation when one uterine artery is the dominant supply of the intervillous flow .The placenta is located laterally in majority of patients with abnormal flow velocity waveforms. In the light of these observations, we designed a prospective study to find out whether the lateral location of placenta as seen by ultrasound at 18-24 weeks of gestation can be used to predict the development of preeclampsia.

For a screening test to be of value, it should be selective, reliably cheap and easy to perform. It should increase the predictive value and the prophylactic measures must be effective.

Good antenatal supervision followed by appropriate treatment will definitely help mother and baby for good outcome.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Pregnancy can induce hypertension in normotensive women (or) aggravate already existing hypertension. Hypertensive disorder in pregnancy continues to take heavy toll of maternal and fetal lives.

Pre-eclampsia, eclampsia remains a difficult puzzle to solve, and it is still in a cloud of mystery.

INCIDENCE

Incidence of pre-eclampsia is commonly cited to be about 5%. Incidence is influenced by parity, race and genetic factors. (Women with positive family history of pre-eclampsia in her mother or in her sister).

In India the incidence of pre-eclampsia amongst hospital patients is about 7-10% of all antenatal admission. In United Kingdom, the incidence is 10%. In USA it is 6-7%. The incidence is found to be higher among nulliparous women, age less than 20 years and more than 35 years.

BLOOD PRESSURE MEASUREMENTS

1. Conventional mercury sphygmomanometer - Gold standard for blood pressure measurement.

2. Use of bell of stethescope as it better amplifies the Korotkoff (K) Sound.

3. Cuff size should be adequate. Bladder length 80% of arm circumference, and width should be 40% of arm circumference.

4. Patient should be in sitting posture with her right arm well supported in a horizontal position at the level of heart and her feet supported (Sibbai).

5. Inflate the cuff above the systolic pressure as recognized by disappearance of the radial pulse.

6. Use of Korotkoff (V) (disappearance of sound) to determine diastolic blood pressure.

CLASSIFICATION

NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAMME WORKING GROUP (2000) (NHBPEP)

I. Gestational Hypertension

Gestational Hypertension or transient Hypertension, Previously called as Pregnancy Induced Hypertension.

- 140/90 mmHg of BP for the first time in pregnancy
- No proteinuria
- Blood pressure returns to normal < 12 weeks postpartum
- Final diagnosis made only postpartum

II. Pre Eclampsia

Occurrence of Hypertension in combination with proteinuria developing after 20 weeks of gestation in a previously normotensive, non proteinuric pregnant women.

It is a pregnancy specific syndrome of reduced organ perfusion secondary to vasospasm and endothelial activation.

Minimum criteria	Increased certainity of Pre eclampsia
• ≥ 140/90 mm Hg>20 weeks of gestation • Proteinuria ≥ = 300mg/24 hrs (or) ≥ = 1 + dipstick	• Blood Pressure ≥ = 160/1l0 mmHg • Proteinuria 2g/24 hours (or) ≥=2+ dipstick • Serum Creatinine 1.2mg/dl unless known to be previously elevated • Platelets < l,00,000/mm^3 • Microangiopathic haemolysis • Elevated ALT/AST • Persistent headache/Other cerebral or visual disturbances • Persistent epigastric pain.

III. Eclampsia

Seizure that cannot be attributed to other cause in a women with pre-eclampsia.

IV. Superimposed Pre-eciampsia on Chronic Hypertension

New onset proteinuria > 300 mg/24 hours in hypertensive women but proteinuria before 20 weeks of gestation.

V. Chronic Hypertension

BP ≥ 140/90 mmHg before pregnancy or diagnosed before 20 weeks gestation not attributable to gestational trophoblastic disease.

Proteinuria

It is directly proportional to maternal and perinatal mortality, (Harth et al 2000) and morbidity.

It is an important sign of pre eclampsia (Chesley 1985). It reflects the glomerular damage that causes leakage of proteins through the basement membrane.

Significant proteinuria is defined by 300mg/24 hours or persistent 30mg/dl (1+dipstick) in random clean catch sample on at least 2 occasions collected 6 hours apart. If proteinuria is >5gm/24 hours (or) persistent 3+ dip stick (or) more the condition is called as severe pre eclampsia (ACOG Practice Bulletin No.33_s January 2002).

Edema

Edema is no longer a part of current definition (or) as a diagnostic criteria of pre- eclampsia (Williams 22nd Edition) and it is seen in nearly 80% of women in last few weeks (Robetson 1971).

Weight Gain

Excessive weight gain of >0.5kg/week because of extracellular fluid volume expansion).

THEORIES REGARDING CAUSATION OF PRE-ECLAMPSIA

IMMUNOLOGICAL

The trophoblastic cells do not express the usual class-I & II MHC antigen, they express unique HLA-G enclosed class-I MHC molecule which allow the trophoblast cells to invade deep into spiral arteries. In patients developing pre-eclampsia there is immune resistance to the invading trophoblast by the maternal immune system, results in poor spiral artery remodeling which is a initially cited feature in pre-eclampsia (ZHOU 1977). Pre-eclampsia is immune mediated, certainly the microscopic changes at maternal placental interface are suggestive of acute graft rejection (Labarrere 1998).

The risk is enhanced when formation of blocking antibodies to antigenic sites in placenta is impaired (Bardegue and Asso, 1991) (or) where the number of antigenic sites is increased as in hyperplacentosis (Beer 1979).

- T helper cells were in a lower proportion in pre-eclampsia (Bardeguez and Associates). T-lympohcyte secrete cytokines that promote implantation and their dysfunction may favour pre-eclampsia (Hayashi & Associates 2004, Whitecar & Collegues 2001).

- Antibodies to B2 glycoprotein I is more relevant immune complex and antiendothelial antibodies may be involved (Taylor etal, 1999).
- Immunocytochemical analysis - VCAM, increased in Pre-eclampsia.
- The pre-eclampsia develops more frequently in multiparous women impregnated by a new Consort (Mostello & Conera 2002, Trapin and Colleagues 1996).

GENETIC SUSCEPTIBILITY

Hereditary estimate of 31% of pre-eclampsia (Nilsson et al)

1. Chesley and Cooper 1986, Pregnancy Induced Hypertension

   is inheritable both by single gene (Broughton Piplus 1999) and Multifactorial inheritance.

   2 Modes

   a) Simple recessive model with genes acting in the mother (gene on chromosome 1, 3, 9)

   b) Dominant model with incomplete penetration

2. Kilpatric reported an association HLA DR4 the transmission from mother to daughter is through HLADR4

3. Angiotensinogen gene variant T235 - high incidence of PIH (Arngrimsson et al)

4. Dizon Townsend and colleagues found high incidence of factor V laden mutation polymorphism gene variant 202109

5. Epigenetic features (or) imprinting is also involved in the pathogenesis of pre-eclampsia susceptibility locus on chromosome lOq 22-1. (Dudejans et al).

6. Hyperhomocystinaemia associated with methyltetrahydrofolate (6778) Homozygozity (Coffer et al).

7. Polymorphism of TNF a and Gene - Mediating endothelial dysfunction, Lymphotoxins, ILi.

8. The eNOS gene susceptibility occurs in the region of chromosome 7q 36.

PLACENTA AND PRE ECLAMPSIA

Many investigators believe that the placenta is the trigger for endothelial cell injury. Evidence suggests that hypoperfused placentas produce various factors that are capable of injuring endothelial cells. Recent data suggest that circulating factors that interfere with the action of vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) play a major role in maternal manifestation of the disorder.

Placental hypoperfusion or ischemia in preeclampsia has many causes. Preexisting vascular disorders such as hypertension and connective tissue disorders can result in poor placental circulation. In cases of multiple gestation or increased placental mass, it is not surprising for the placenta to become underperfused. However, most women who develop preeclampsia are healthy and do not have underlying medical conditions. In this group of women, abnormally shallow placentation has been shown to be responsible for placental hypoperfusion.

The shallow placentation noted in preeclampsia is a result of the inability of trophoblasts to invade the decidual vessels. In normal pregnancies, a subset of cytotrophoblasts called invasive cytotrophoblasts migrate through the implantation site and invade

decidua tunica media of maternal spiral arteries and replace its endothelium in a process called pseudovascularization. As a result of these changes, these vessels undergo transformation from small muscular arterioles to large capacitance, low-resistance vessels. This allows increased blood flow to the maternal-fetal interface. Remodeling of these arterioles probably begins in the first trimester and ends by 18-20 weeks' gestation. However, the exact gestational age at which the invasion stops is unknown.

In preeclampsia, this invasion of the decidual arterioles is incomplete. The invasive cytotrophoblasts fail to replace tunica media, resulting in mostly intact arterioles that are capable of vasoconstriction. Histologic evaluation of the placental bed demonstrates few cytotrophoblasts beyond the decidual layer.

The trophoblast differentiation along the invasive pathway involves alteration in the expression of a number of different classes of molecules, including cytokines, adhesion molecules, extracellular matrix, metalloproteinases, and the class Ib major histocompatibility complex molecule, HLA-G Angiogenesis is critical for successful placentation. Both VEGF and PlGF promote angiogenesis by interacting with the VEGF receptor family.

While both growth factors are produced by placenta, the serum level of PlGF rises much more significantly in pregnancy. Taylor et al demonstrated that the serum level of PlGF decreased in women who later developed preeclampsia. The fall in serum level was notable as early as the second trimester in women who developed preeclampsia and intrauterine growth restriction.

In 2003, Maynard et al observed that the serum levels of both VEGF and PlGF were decreased in women with preeclampsia. However, the magnitude of decrease was less pronounced for VEGF since its serum level was not as high as PlGF, even in normal pregnancy. Others have confirmed this finding and showed that the serum level of PlGF decreased in women before they developed preeclampsia.

Khong et al showed that poor trophoblastic conversion of spiral arterioles leads to development of pre eclampsia. Both uterine arteries have a significant number of branches. When placenta is laterally located the uterine artery close to the placenta has lower resistance than the one opposite from it. In patients with centrally located placentas both uterine arteries demonstrate similar resistance.

It is possible that when the placenta is centrally located, the uteroplacental blood flow needs are met by equal contribution from both the uterine arteries. However when the placenta is laterally located., in the majority of patients the uteroplacental blood flow needs are to be met primarily by one of the uterine arteries, with some contribution by the other side via collaterals. This collateral contribution may be deficient in some individuals facilitating development of pre eclampsia.

ENDOTHELIAL DYSFUNCTION

Endothelial damage account for all aspects of pathophysiology of pre-eclampsia (Roberts et al 1998).

Endothelial dysfunction results from "Generalised perturbation of normal maternal intravascular inflammatory adaptation to pregnancy (Redman and Colleagues (1999).

Immunologically mediated deficiency in trophoblastic invasion of spiral arteries results in release of a number of factors into maternal circulation. These changes inturn provoke activation of vascular endothelium (Hayman and Asso., 2000, Ness and Roberts 2000, Walker 2000). Damaged endothelium activates endothelial cells to promote coagulation and increases sensitivity to vasopressor agents.

Reduction of unbound serum level of Angiogenic factors such as VEGF and PLGF due to upregulation of soluble fms like tyrosine kinase receptor (Maynard et al) and Serum level of sFltl is increased in pre-eclampsia leads to endothelial dysfmction.

Prostaglandin theory (Wang et al 1991): In pre-eclampsia, the balance is between PGI2 & TXA2 (Prostocycline) (Thromboxane A2) is titled so TXA2 is found to increased (Walsh 1985).

ENDOTHELINS

They are polypeptides with potent vasoconstricting property, higher level of Endothelin I is found in pre-eclampsia (Clark Schiffl992).

NITRIC OXIDE

Decreased in pre-eclampsia (Anumba Ford Ant obsgyn 181: 14 (1999). This concept is disproved. Decreased endothelial nitric oxide synthase expression (Wang & Collagen 2004). Increased production of Nitricoxide as a compensatory mechanism (Benedetto & Asso., 2000). It is a consequence of hypertension, but not the cause (Morris and Colleagues 1996).

Ionic calcium is crucial for synthesis of vasosubstances in the endothelium like PGI2 and Nitricoxide. An alteration in the action of Nitricoxide may be related to inactivation by free radical superoxide (secondary to inflammatory process) (Lopez et al).

OXIDANTS AND ANTIOXIDANTS

A work by Hubal et al have confirmed that pre-eclampsia may have its origin in a disturbed oxidation mechanism. Increased free radical in Pre-eclampsia (Wisdom and Ass., 1991), free radical causes endothelial damage.

PLACENTAL PROTEINS

Corticotrophin releasing factor, HCG, Activin A & inhibin A are said to play a role in pathogenesis of Pregnancy induced hypertension.

VASOSPASM

Vasospasm that characteristic of the disease (Volhard). Vasopasm causes endothelial damage and interendothelial cell leakage which results in subendothelial deposition of fibrin and platelets and hence uteroplacental insufficiency (Brunner & Gavras 1975).

SCREENING TEST

Comparison of various other screening tests with urinary calcium/ Creatinine ratio as a predictor of Pre-eclampsia.

A variety of biochemical and biophysical markers based primarily on rationales implicated in the pathology and pathophysiology of pre-eclampsia have been proposed for its prediction.

The good screening test should be sensitive, cheap, easy to perform, and readily interpretable with high predictive value.

Screening test can be divided into 3 categories.

I. Haemodynamic Tests

1. Angiotension II infusion test: Talledo et al 1968

Abnormal vascular reactivity of patients destined to develop pre-eclampsia may be detected several weeks before the clinical signs and symptoms appears. Women requiring less than 8ng/kg/minute of Angiotension II to raise their diastolic BP by 20mmHg had a positive predictive value of 20-40% of developing pre-eclampsia (Friedman).

2. Isometric hand Grip Test

Dagani et al Increase in diastolic pressure of more than 20mmHg during a hand grip exercise test at 28-32 weeks had positive predictive value of 20-30%. This test is not affected by positional change and is safe and easily performed, although it is time consuming taking upto 30minutes to perform. Since hand grip represent sympathetic nervous system activity. There is little evidence that pre-eclampsia is mediated by sympathetic activity. This requires further evaluation. False negative 4% & False positive 19%.

3. Roll over test

Proposed by Gant et al, An increase > 20mmHg of diastolic blood pressure induced by having women assume the supine position after lying left lateral position done at 28 - 32 weeks was shown to be associated with later on occurrence of pre-eclampsia is 33%. Though the test, is simple to perform and requires only time and personnel rather an elaborate equipment.

4. Mean arterial pressure - Page and Christianson

Suggested that patients with MAP of >90mmHg in II trimester should be regarded as a risk category but the predictive value varies

greater from one study to another. The position of the arm relative to heart level also affect the recording. There is also variation of blood pressure with the circadian rhythm, values being highest during the afternoon and early evening. This method to be effective, increased uniformity of recording measurement is necessary.

5. Uterine artery Doppler Velocimetry

Doppler measurement of uterine artery impedance in the mid II trimester (18-24 weeks) is an early screening test (Bewley, 1991), Chappel based on the presumption that impaired trophoblastic invasion of spiral arteries causes decreased utero placental blood flow. The presence of a high systolic - diastolic ratio, persistence of diastolic notch may predict pre-eclampsia (Irion et al 1998) and Resistance Index > 0.58. Positive predictive value was only 28% (Friedman and Lindheimer 1999). Studies concluded that the test was not helpful in the management of individual patients.

6. Placental Position

URINARY ASSAYS

1. Urinary calcium excretion

Studies found that urine calcium excretion is reduced in pre-eclampsia. A study showed that urinary calcium excretion average of 313+140mg in normal pregnancy and 248+134 in transient hypertension. Hypocalciuria is due to increased distal tubular reabsorption alternatively proximal tubular reabsorption. 24 hour urinary calcium excretion less than 12 mg/ dl had sensitivity of 88% and positive predictive value of 91%. (Sanchez - Romos, Obstet & Gynecol 1991).

Another study reported that patient with pre-eclampsia had significantly less excretion of total calcium (129.7 + 18.7mg/24 hours) than normotensive (283.9 + 12.3) or those with gestational hypertension (233.2 + 22.3) (P = 0.0001) using a receiver operator curve, urinary calcium threshold of 12mg/ dl was choosen as a predictor for the development of pre-eclampsia with sensitivity of 83% specificity of 91%, Positive predictive value 83, Negative predictive value 91.

2. Urinary calcium / Creatinine Ratio

A number of studies have shown that a low calcium and creatinine excretion is a valuable marker for pre-eclampsia prediction. Since creatinine clearance is not significantly different among normotensive and pre-eclampsia, (134.4 + 14.9) in Pre-eclampsia (141.9 + 7.2) and in gestational Hypertension, (150.2+6.5) in normotensive pregnant women. Studies were done using single voided urinary calcium / creatinine ratio in the prediction of pre-eclampsia and it correlated well with 24 hours calcium excretion. Hence 24 hours urinary calcium excretion can be estimated from single voided urine sample.

3. Micro albuminuria

By Radio immuno assay can detect microalbuminuria of the value >11 ug/ml when done between 24-34 weeks it indicates positive result.

4. Urine Kallikrein / Creatinine ratio

Kallikrein Creatinine ratio of <170 between 16 and 20weeks of pregnancy predicts future development of pre-eclampsia. Camphell et al., 1987: sensitivity of 83% false positive ratio of 50%, false negative ratio 10%, positive predictive value of 91%.

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5. **Urinary metabolites of PGI_2** - help in diagnosis of pre-eclampsia

6. Markers for endothelial dysfunction.

Fibronectin, a glycoprotein synthesized in the vascular endothelium elevated in pre-eclampsia - 2 fold increase in fibronectin, >400 ug/ml. Positive predictive value was only 39% negative predictive value 98% (Chavarria et al, 2003) Sensitivity is quiet low only (69%) (Pallberg & Colleagues).

Plasminogen inhibitor 1 is increased relative to plasminogen activator (Caron and Colleagues 1991) Thrombomodulins, cell adhesion molecules and Endotheline I are also found to be increased.

7. Coagulation factors

1) Increased Thromboxane A_2 (Fitzgerald et al). 2) Increased Factor VIII. 3) Decreased Antithrombm III. 4) Thrombocytopaenia and abnormalities platelet function (Aggregation).

8. Cytokines

Increase IL and TNF a - not yet proved sufficiently (Savvidon and colleagues 2002, Benyo 2000).

9. Anti angiogenic factor

Levin RJ et al, soluble fms like Tyrosine Kinase I, Endoglin-Increased, Ratio of sFltl: PIGF - more accurate (Devivo et al).

MARKERS OF OXIDATIVE STRESS

1. Malondialdehyde lipid peroxidation (Hubel & Co-authors 1989).

2. Pro oxidants (Herbert and Colleagues 1994)

3. Homocysteine (Cotter and Associates 2001) elevated level around mid pregnancy had a 3-4 fold risk of development of pre-eclampsia.

4. Triglycerides, free fattyacids and lipoprotein (Hobel and Colleagues 1996).

5. Prostaglandin Isomerase; marker of impending pre-eclampsia (Regan and Fitz Gerald, 1997).

These tests are not economical they are not popularized.

OTHERS

1. Uric Acid

Elevated uric acid levels exceeding 5.9 mg/dl is considered significant correlation well with sensitivity and perinatal outcome.

2. Fibronectin

Increased serum cellular fibronectin level in some women with pre-eclampsia (Magann EF et al).

3. Placental peptides

HCG (Ashoc R & colleagues 1997) inhibin A & inhibin B - in the search for early pregnancy marker for pre-eclampsia (Woodruff)

4. Plasma P Selectin

Plama P selectin as the earliest predictor in the first trimester is under study.

5. Urinary podocyte excretion

Highly sensitive and specific marker (Garovic et al).

6. Enzymes and Hormones:

Increased levels of plasma cystyl amino peptidase, Pappa-A (Wood and Durham) and high level of (3 HCG (Noguera et al).

7. Foetal DNA

Foetal DNA in maternal serum may be predictive of pre eclampsia.

PREVENTION OF PRE-ECLAMPSIA

Pregnancy is a process of physiologic adaptation which occur primarily is an effort to supply the developing fetus with their nutrients that are essential for its proper development and growth.

Preventive measures have been concentrated to relieve vasospasm and to correct the disturbed prostaglandin synthesis which lead to platelet aggregation and endothelial damage. There are no definite preventive methods but attempt should be made for early detection of high risk patients.

Thomas Brewer rightly stated that pre-eclampsia is a complication of maternal malnutrition whose symptoms can be largely eliminated by eating a well balanced diet.

A study by the ***Dietary Approaches to stop Hypertension (Dash)*** demonstrated that dietary manipulation significantly lowers the Blood pressure.

Prevention can dealt with

1. Non medical measures
2. Medical measures.

SALT RESTRICTION

This may be used as a means to prevent the onset of essential hypertension but not be used for gestational Hypertension and Pre-eclampsia.

BED REST

This may be useful in known hypertensive patients to reduce the severity.

FISH OIL

The benefit from fish oils seems to be associated with long chain n-3 fatty acids, C-20, n3 eicosepentoicacid and docosepentanoicacid & docosahexanoid acids. They decrease platelet aggregation on endothelial cells and leads a relative state of vasodilatation.

PLANT OIL

Linoleic and Evening primrose oil (3mg Linoleic + 32mg of Gammalinoleic acid / day.

CALCIUM

Proposed mechanism where by calcium supplementation may reduce blood pressure.

Reduced serum calcium

↓

Increases Vit. D stimulation - interact with renin / angisotensin

↓

(Through its link to PTH) Vit. D receptors present in the smooth muscle reduction of altered Vit. D metabolism smooth muscle proliferation

↓

Development of atherosclerosis

↓

Hypertension

High intake of calcium of L.3g/d showed a reduced incidence of both Pre-eclampsia & Eclampsia (Villar, J American J Clin -Nutrition 1980). World Health Organization (2006) supplementation with calcium 1.5g/day significantly reduces the risk of maternal neonatal morbidity and preterm delivery in the later among young women.

Cochrane systematic review noted protective effects of calcium supplementation only in women with low calcium intake.

ANTIOXIDANT

Antioxidant therapy significantly reduced endothelial cell activation in a study performed by (Chappell and associates, Lancet

1999). There was a significant reduction in the incidence of Pre-eclampsia in women who was given with vitamin C and E.

MEDICAL MEASURES

Low dose aspirin appears to be beneficial for women at high risk of pre-eclampsia (Cochrane Review - 19% reduction in risk of pre-eclampsia).

AIM OF THE STUDY

AIM OF THE STUDY

To analyse whether placental location as determined by ultrasound at 18-24 weeks can predict the subsequent development of pre-eclampsia.

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was conducted at department of Obstetrics and Gynaecology, Govt. R.S.R.M. Lying in Hospital attached to Stanley Medical College, Royapuram, Chennai.

STUDY PERIOD: The Period of Study was from July 2009 to August 2010.

STUDY DESIGN: PROSPECTIVE STUDY

300 Pregnant women attending the antenatal clinic at R.S.R.M. Lying in Hospital, were registered in this study. Only those pregnant women whom we could follow to term and were planning delivery at R.S.R.M. Lying in Hospital, Royapuram Chennai, were included in this study.

The location of the placenta was determined by ultrasound at 18-24 weeks in all the 300 women. The placenta was classified as central when it was equally distributed between the right and the left side of the uterus irrespective of anterior, posterior or fundal position. When 75% or more of the placental mass was to one side of the midline, it was classfied as unilateral right or left placenta. BP was

measured in every subsequent visit. The end point of the study was the development of hypertension as per the ACOG criteria or delivery.

PATIENTS SELECTION CRITERIA

Inclusion Criteria

1. Gestational age between 18-24 weeks.
2. Women who intend to have their deliveries at RSRM lying in hospital.

Exclusion Criteria

1. History chronic hypertension
2. History of Diabetes Mellitus
3. History of Renal disease
4. Blood pressure > 140/90 mm Hg
5. Evidence of proteinuria by the dipstick method.

The study was done on asymptomatic primi, and pregnant women with Rh Negative, with previous h/o pre-eclampsia, family h/o pre-eclampsia were also included.

METHODOLOGY

In all the pregnant women included in the study, a written informed consent was obtained.

1. No dietary alterations were recommended

2. Detailed history was taken.

3. Complete examination

    i. General examination

    ii. Clinical examination

        - Cardiovascular system
        - Respiratory system
        - Central nervous system

    iii. Obstetric examination was done.

4. Blood Pressure

    i. In sitting posture

    ii. Phase Korotkoff V sound was taken to determine the diastolic component

5. Basic Investigations

i. Haemoglobin

ii. Blood Grouping and Rh Typing

iii. Urine-Albumin

- Sugar

- Deposit

6. The location of the placenta was determined by ultrasound at 18-24 weeks in all the 300 women. The placenta was classified as central when it was equally distributed between the right and the left side of the uterus irrespective of anterior, posterior or fundal position.

When 75% or more of the placental mass was to one side of the midline, it was classified as unilateral right or left placenta. The end point of the study was the development of hypertension as per the ACOG criteria or delivery.

7. Followed up with routine antenatal visits for signs and symptoms of pre-eclampsia by routine examination of blood pressure, serial weight, edema, and investigation of pre-eclampsia when required and results were tabulated.

8. Mode of delivery and fetal outcome was noted

RESULTS

RESULTS

TABLE-I

DISTRIBUTION OF PERSONS IN STUDY GROUP

ACCORDING TO AGE

<20 yrs	20-25 yrs	25-30yrs	>30 yrs
63	152	63	22
21%	50.7%	21%	7.3%

50.7% of mothers were in the 20-25 years age group. 21% in <20 years age group. Another 21% in 25-30 years age group. 7.3% in the greater than 30 years age group.

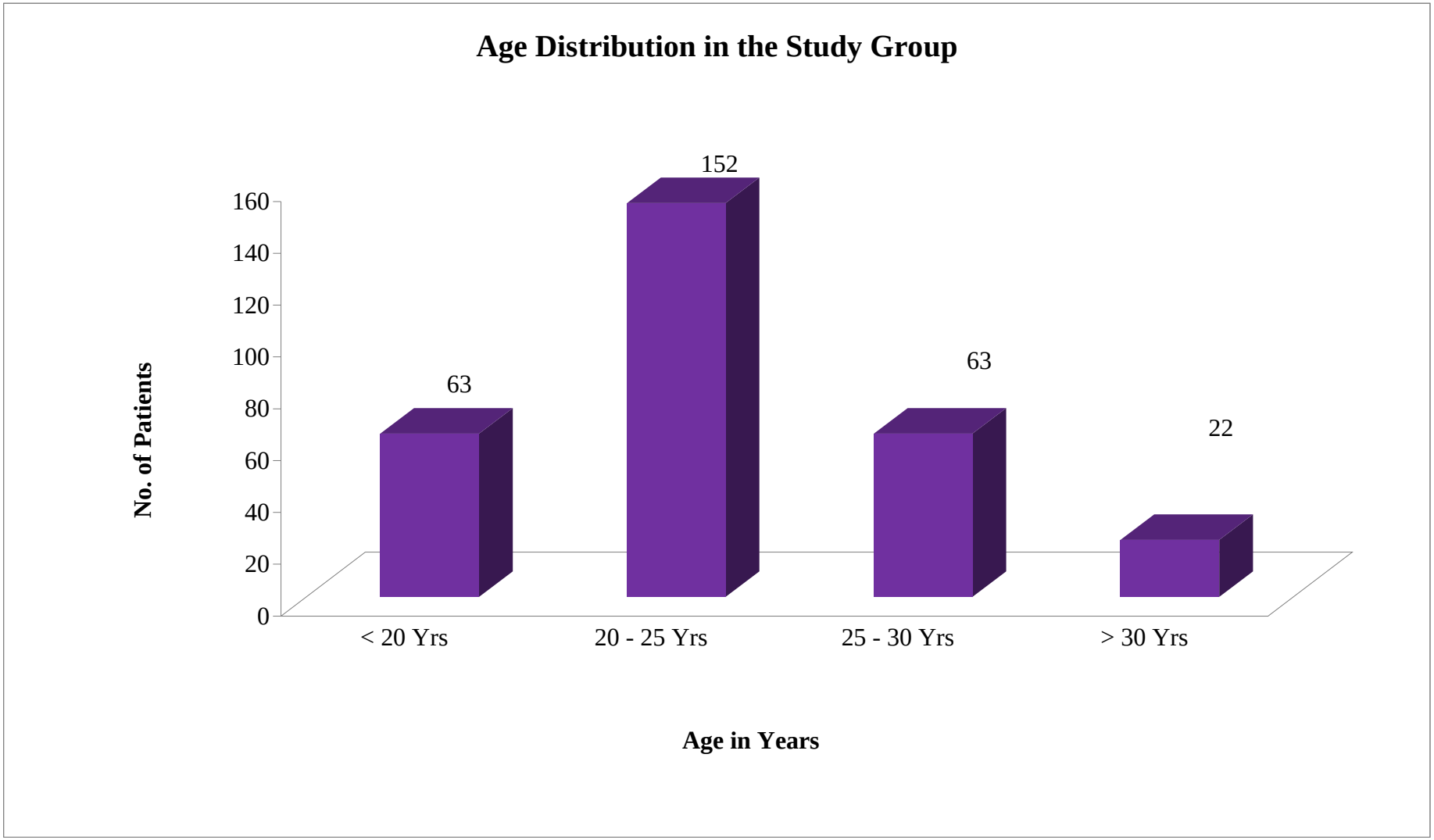
Age Distribution in the Study Group
No. of Patients
160
140
120
100
80
60
40
20
0
63
152
63
22
< 20 Yrs
20 - 25 Yrs
25 - 30 Yrs
> 30 Yrs
Age in Years

TABLE-II

DISTRIBUTION OF PATIENTS WITH PRE-ECLAMPSIA ACCORDING TO AGE

<20 years	20-25 years	25-30years	>30years
4	26	8	4

The relationship of maternal age and incidence of pre-eclampsia shows higher incidence among young primi gravida and markedly increased incidence among older primigravida. In study group preedampsia was high between 20-25 years and pre-eclampsia was high above 30 years.

Distribution of Patients with pre eclampsia according to age

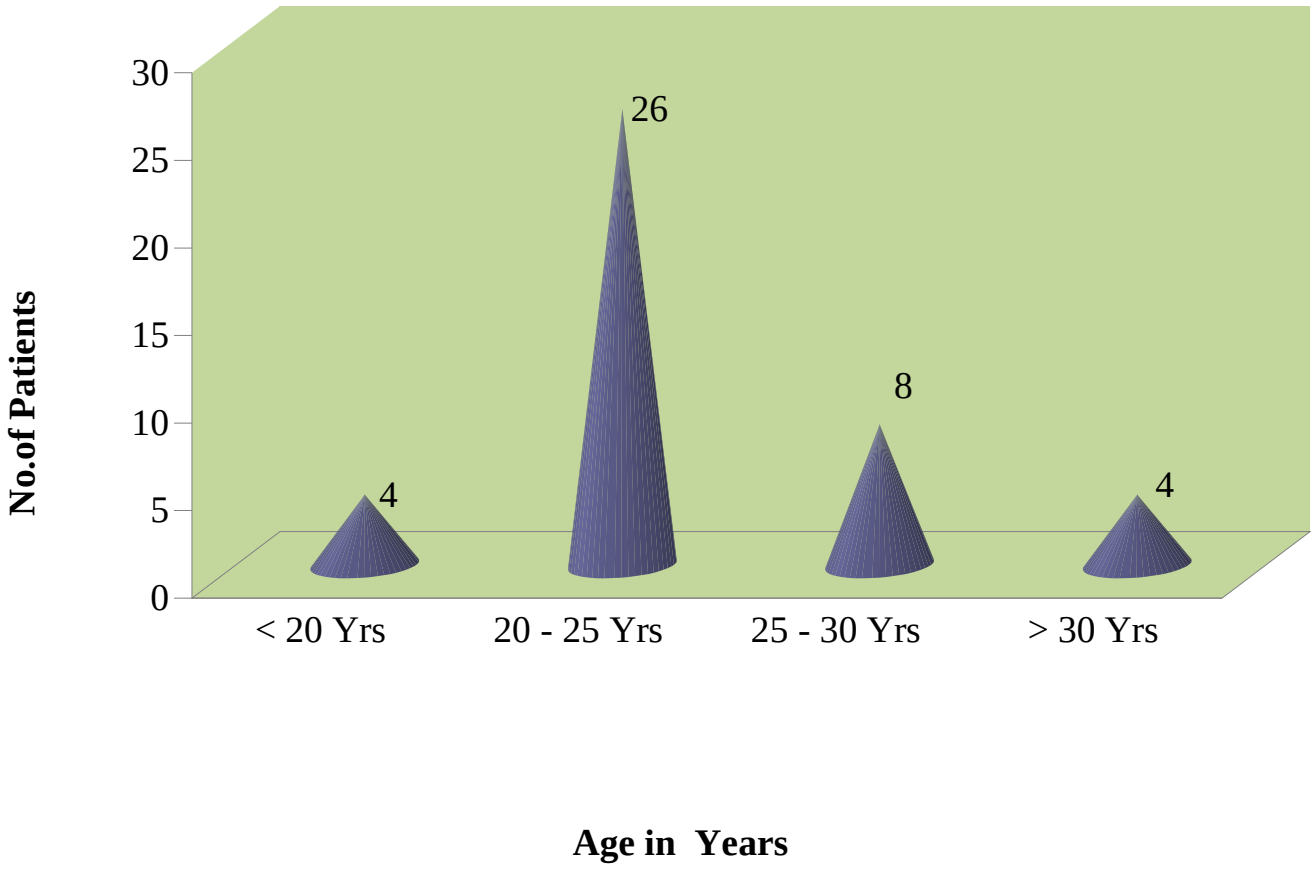

TABLE-III

DISTRIBUTION OF PATIENTS WITH PRE-ECLAMPSIA ACCORDING TO PARITY

PARITY	TOTAL	PRE-ECLAMPSIA POSITIVE	%
Primigravida	222	34	81%
Multigravida	78	8	19%

"Above table shows the distribution of patients with pre-eclampsia according to parity. Pre eclampsia is more common in primigravida. This table show high incidence of pre-eclampsia in primigravida (81%) than in multigravida.

Distribution of Patients with pre-eclampsia according to parity

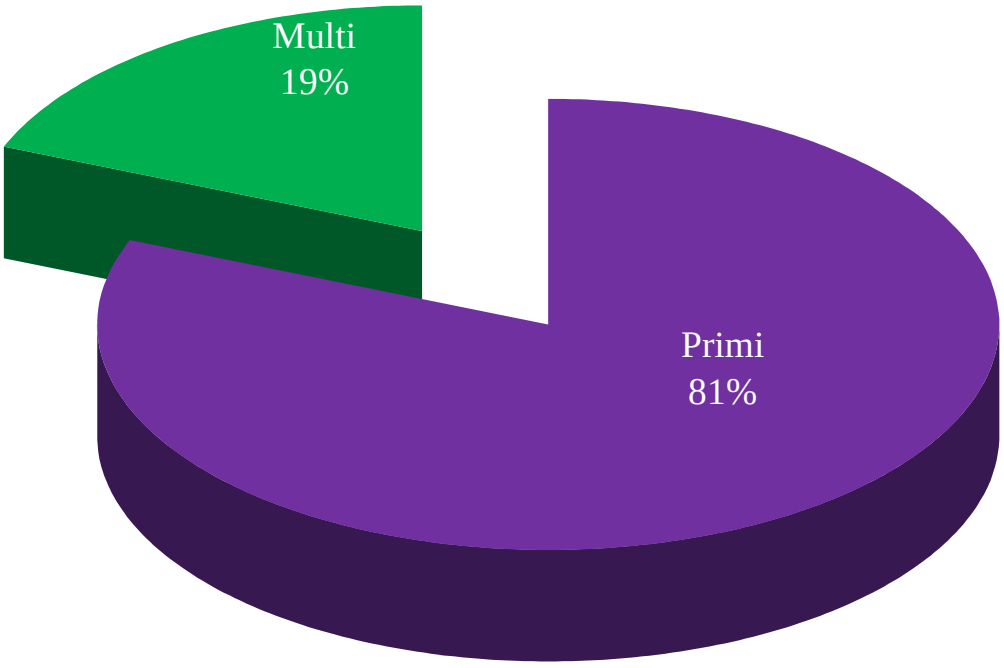

TABLE-IV

RELATIONSHIP OF PLACENTAL POSITION AND DEVELOPMENT OF PRE-ECLAMPSIA

PLACENTAL POSITION	DEVELOPED PRE ECLAMPSIA	NORMOTENSIVE
CENTRAL (228)	8(19.1%)	220(85.2%)
LATERAL(72)	34(80.9%)	38(14.8%)

This table shows the relationship of placental position and development of pre-eclampsia. 34 patients in the lateral placenta group developed pre eclampsia which accounted to 81% of all pre eclamptics.

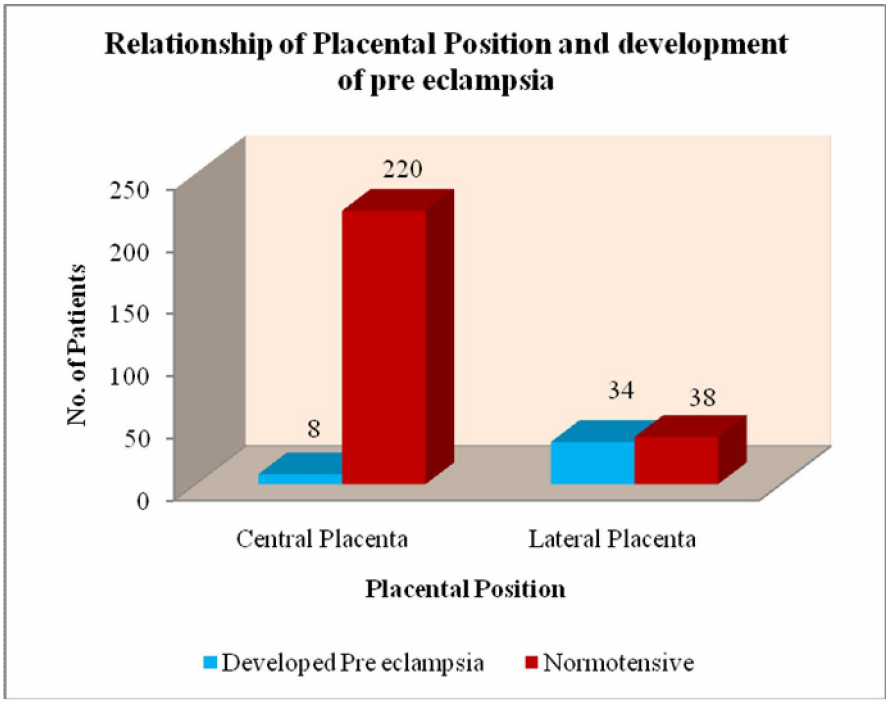
Relationship of Placental Position and development of pre eclampsia
No. of Patients
250
200
150
100
50
0
220
8
34
38
Central Placenta
Lateral Placenta
Placental Position
Developed Pre eclampsia
Normotensive

TABLE-V

RELATIONSHIP OF PLACENTAL POSITION AND DEVELOPMENT OF PRE-ECLAMPSIA IN HIGH RISK WOMEN

PLACENTAL POSITION	DEVELOPED PRE ECLAMPSIA	NORMOTENSIVE
CENTRAL (53)	5(33.3%)	48(88.9%)
LATERAL(16)	10(66.7%)	6(11%)

This table shows the relationship of placental position and development of pre-eclampsia in the high risk group. 10 patients in the lateral placenta group developed pre eclampsia which accounted to 66.7% of all pre eclamptics in the high risk group.

p Value is 0.001 and is highly significant.

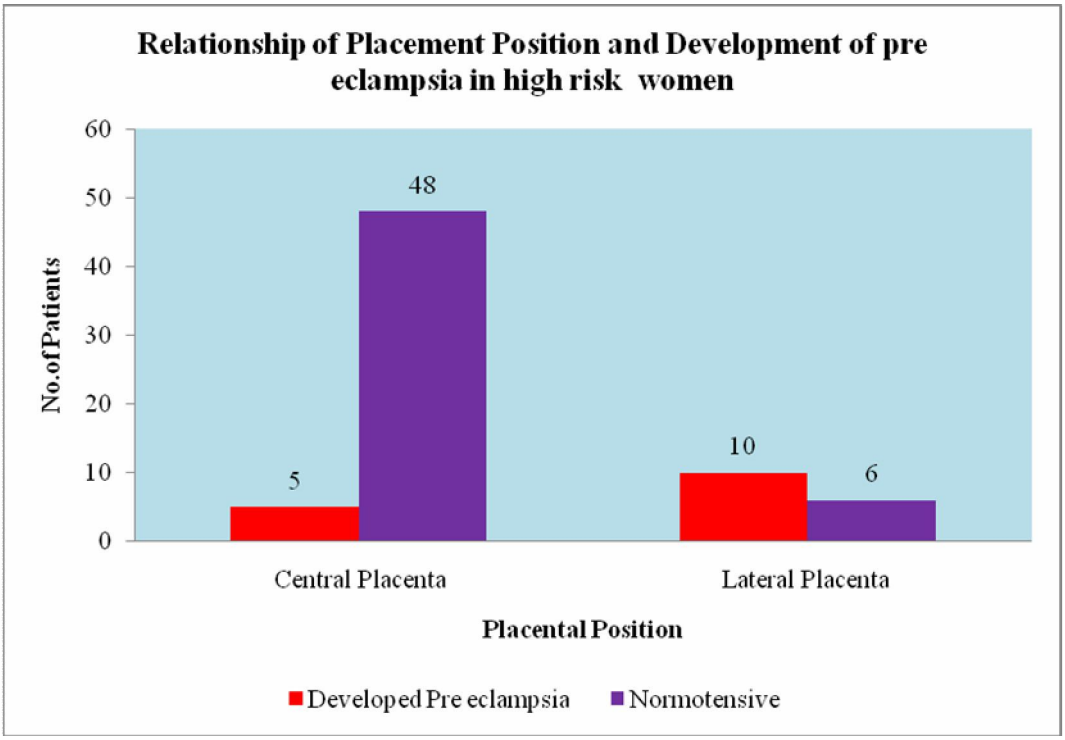
Relationship of Placement Position and Development of pre eclampsia in high risk women
No.ofPatients
60
50
40
30
20
10
0
48
5
10
6
Central Placenta
Lateral Placenta
Placental Position
Developed Pre eclampsia
Normotensive

TABLE-VI

RELATIONSHIP OF PLACENTAL POSITION AND DEVELOPMENT OF PRE-ECLAMPSIA IN LOW RISK WOMEN

PLACENTAL POSITION	DEVELOPED PRE ECLAMPSIA	NORMOTENSIVE
CENTRAL (175)	12(44.4%)	163(79.9%)
LATERAL(56)	15(55.6%)	41(20.1%)

This table shows the relationship of placental position and development of pre-eclampsia in the low risk group. 56% of the pre eclampsia patients in this group had lateral placenta.

Relationship of Placental Position and development of pre-eclampsia in low risk women

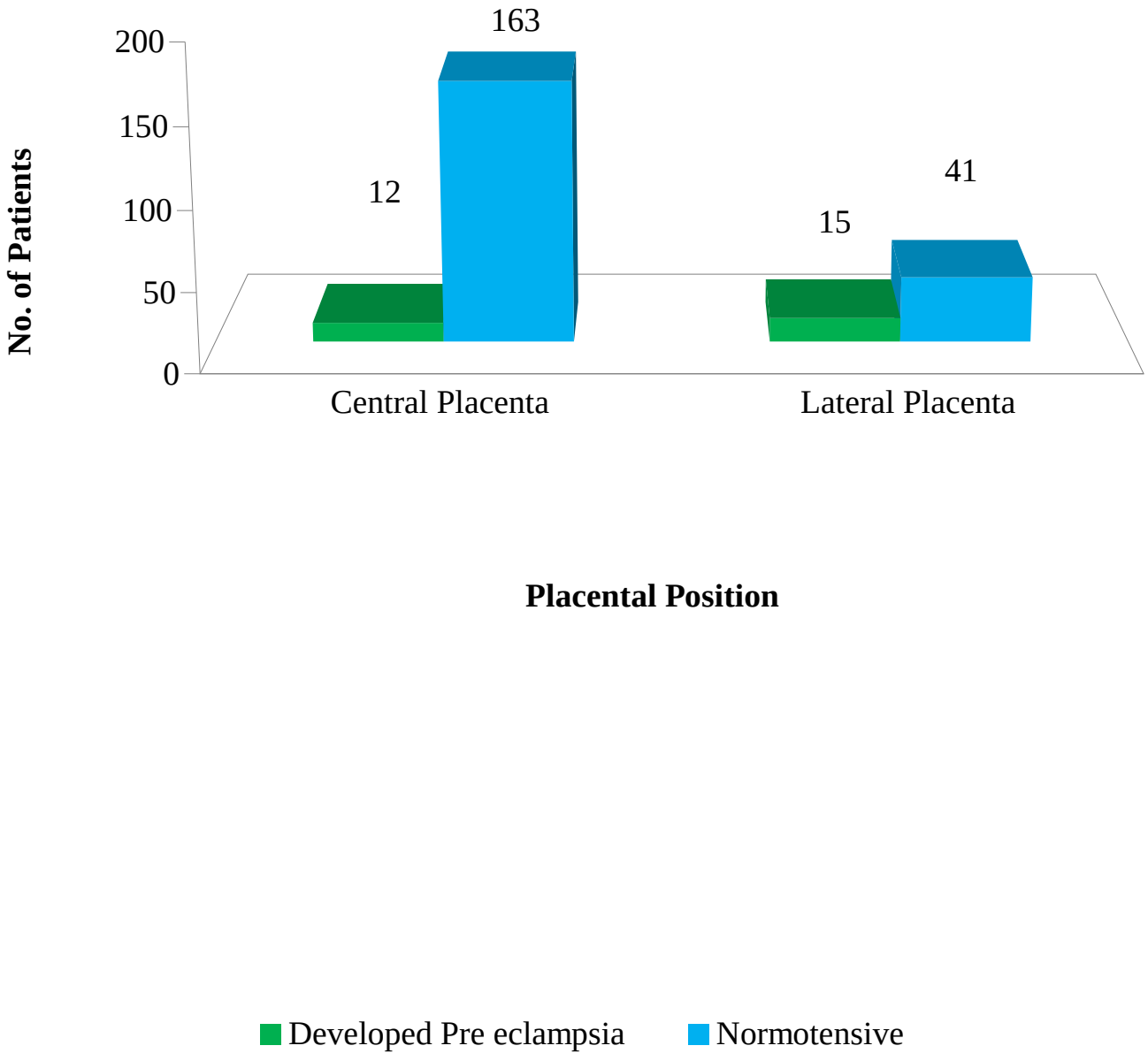

TABLE-VII

DISTRIBUTION OF PATIENT ACCORDING TO THEIR FIRST APPEARANCE OF PRE ECLAMPSIA IN WEEKS

Gestational age in weeks	Number of Patients
26-28	1
29-31	3
32-34	6
35-37	15
38-40	17
>40	-

The above table shows most patients developed pre-eclampsia between gestational age of 36 to 40 weeks indicating that the incidence is higher in later part of gestation

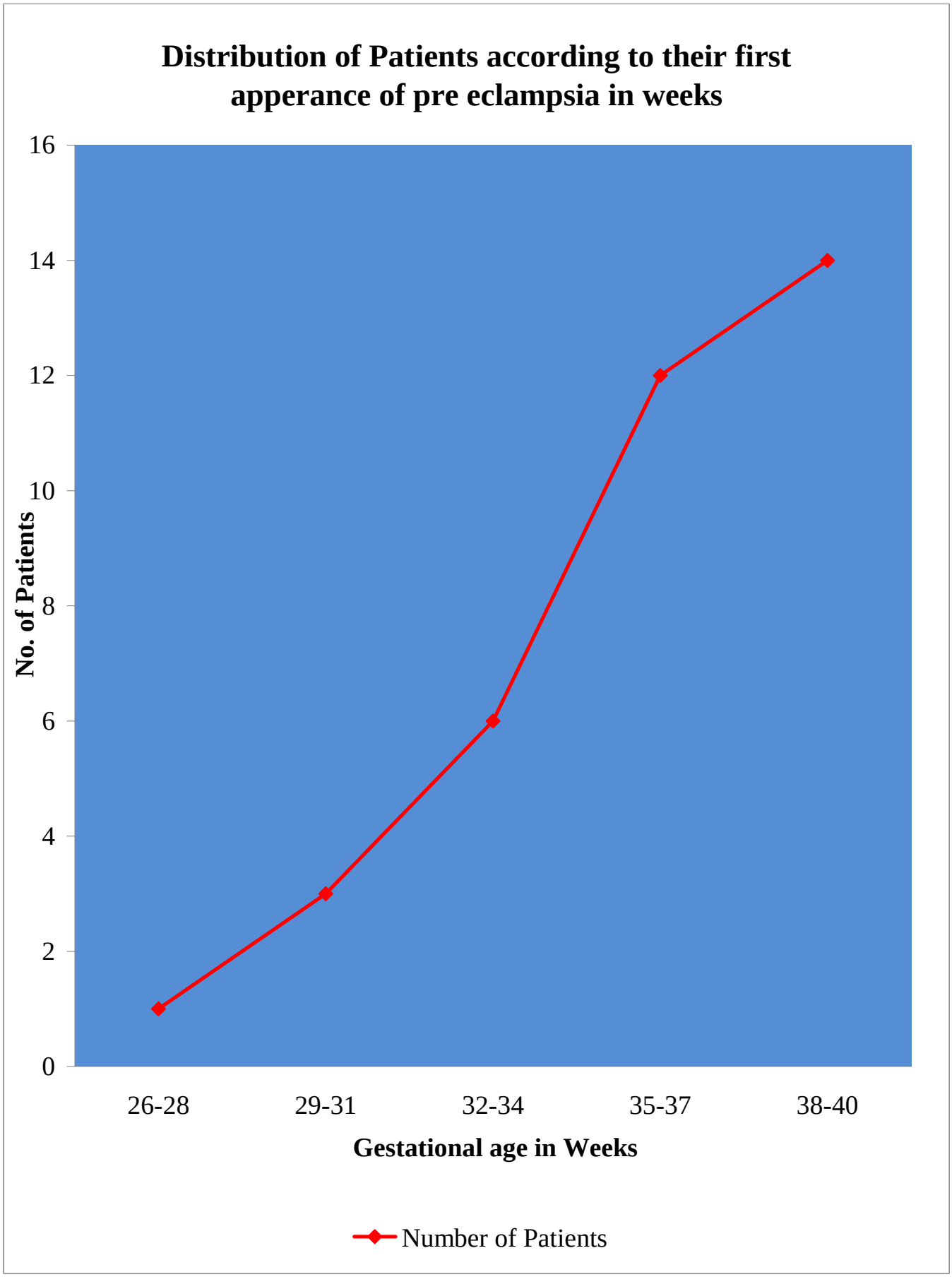
Distribution of Patients according to their first apperance of pre eclampsia in weeks
16
14
12
10
8
6
4
2
0
No. of Patients
26-28
29-31
32-34
35-37
38-40
Gestational age in Weeks
Number of Patients

TABLE-VIII

DISTRIBUTION OF PATIENTS ACCORDING TO SEVERITY OF PRE-ECLAMPSIA IN STUDY AND CONTROL GROUP

Type of pre-eclampsia	Study group
Mild Pre-eclampsia	38
Severe Pre-eclampsia	4
	42

The above table denotes that distribution of persons in study group according to severity of pre-eclampsia. 38 patients shad mild pre-eclampsia and 4 patients in the study group had severe pre-eclampsia.

TABLE-IX

PREDICTIVE VALUE OF PLACENTAL POSITION IN PREDICTING PRE ECLAMPSIA

PLACENTAL POSITION	DEVELOPED PRE ECLAMPSIA	NORMOTENSIVE
CENTRAL (228)	8(19.1%)	220(85.2%)
LATERAL(72)	34(80.9%)	38(14.8%)

This screening test has

- Sensitivity of 81%
- Specificity of 85.3%
- Positive predictive value of 47.2%
- Negative predictive value of 96.4%
- p value < 0.001 which is significant.

DISCUSSION

DISCUSSION

The study was conducted on asymptomatic pregnant women between 18-24 weeks of gestation. All women included in the study were normotensive with a BP lessthan 140/90 mmHg.

Placental location was determined by realtime ultrasonogram. Placenta was classified as right or left lateral regardless of its antero posterior position. For purpose of analysis the patients with right or left placentas were classified as having unilaterally located placentas.

In this study 300 patients were registered. Out of which 231 were in the low risk pregnancy group and 69 in the high risk pregnancy group. Of the 300 patients 42 developed pre eclampsia with incidence of 14%.

INCIDENCE

Mudaliar & Menon	10%
Sanchez Ramos	14%
Ritu Kamara et al	13.6%
Rodriguesz et al	10.6%
Present study	14%

Present study shows 14% incidence of pre eclampsia which is similar to Sanchez Ramos study.

AGE INCIDENCE

Women of different age group were included in the study and control group. 50.7% of those who developed pre-eclampsia were in the age group of between 20-25 years. This is accordance with MACGILLVIRAY'S report on age incidence of Pre-eclampsia which states that the incidence of pre-eclampsia is high among young primigravida. 7.3% of those who developed pre-eclampsia were in the age group more than 30 yrs. Observation showed that the extremes of age group has predilection for pre-eclampsia.

PARITY

The incidence of pre-eclampsia is high in primigravida when compared to multigravida .The overall incidence of pre-eclampsia in this study was 14% The overall incidence of pre-eclampsia inprimigravidae in this study was 15%.

The incidence of pre-eclampsia in primigravida quoted in different studies.

Long & Oats	14.1%
Norwitz et al.,	11.9%
Williams	7.6%
Present study	**14%**

Our study showed results similar to the study by Long and Oats.

Of the 300 patients enrolled 231 were in the low risk pregnancy group. Totally 42 women developed pre eclampsia- incidence of 14%. In all 228 women had central placenta and 72 women had lateral placenta. Only 19% (8 women) with central placenta developed pre eclampsia. But in the lateral placenta group 80.9% developed pre eclampsia of which 4 had severe pre eclampsia. 1 woman in this study developed eclampsia nbecause of poor compliance.

In high risk group, 69 women-

- 14 patients had GDM,
- 13 patients- BOH,
- 3-treated for infertility and
- 42 patients had medical disorders like Bronchial Asthma, Epilepsy, heart diseases, anaemia, hematological disorders, thyroid dysfunction and Rh negative.

Of these 53 women had central placenta and 16 women had lateral placenta. In the lateral placenta group 67% developed pre eclampsia.

In low risk group with 231 women, 175 had central placenta and 56 had lateral placenta. In the lateral placenta group 56% developed pre eclampsia.

My study has a sensitivity of 81% in predicting pre eclampsia, and specificity of 85.3%. The positive predictive value of this screening test is 47.2% and so it is for various other screening tests.

Comparison of predictive value of placental laterality in present study with other studies

Author	**Year**	**No of patients**	**Sensitivity in%**	**Specificity in %**	**PPV in %**	**NPV in %**
Muralidahar Pai et al	2005	426	73	86	51	94
Alexander Kofinas etal	1989	153	79	81	46	90
Ozcan et al	1996	182	72	80	52	94
Present Study	2010	300	81	85.%	47.2%	96.4

COMPARISON OF OUR STUDY WITH OTHER STUDY

Our study shows a sensitivity of 81% correlating with Kofinas et al study. Our specificity of 85.3% correlated well with Muralidhar Pai's study.

Our positive predictive value of 47.2% correlated with Kofinas et al study and negative predictive value of 96.4% correlated well with Muralidhar Pai and Ozcan et al studies. Thus placental position is an easy non invasive test to predict pre eclampsia.

SUMMARY

SUMMARY

The results of analysis of the study is summed up as follows:

1. Pre-eclampsia developed in 14% of patient population between 36-40 weeks of gestation mostly. Patients developed pre-eclampsia in the form of raised blood pressure, proteinuria and with or without odema.

2. Nulliparity was found to be a significant high risk factor.

3. 47% patients in the lateral placenta group developed pre eclampsia. Only 3.5% in the central group developed pre eclampsia.

4. 80.9% of the pre eclampsia patients had lateral placenta and only 19.1% had central placenta.

5. Sensitivity – 81%, specificity – 85.3% , positive predictive value - 47.2%, and negative predictive value of 96.4% which is significant.

CONCLUSION

CONCLUSION

The study shows that placental position determined by ultrasonogram between 18-24 weeks of gestation is an excellent screening tool for the prediction of pre-eclampsia among numerous screening test with specificity of 85.3% and sensitivity of 81% and positive predictive value 47.2%, and negative predictive value 96.4%.

This test is ideal because

- Simple and easy to perform
- Inexpensive and part of the anomalies scan performed.
- Non invasive and convenient for the patient

Lateral placentation helps identify the population who is at greatest risk to be included in primary preventation program.

PROFORMA SHEET

NAME: AGE: IP NO: UNIT:

ADDRESS :

DISSERTATION REF No:

EDUCATIONAL STATUS :

SOCIO-ECONOMIC STATUS :

MENSTRUAL HISTORY: L.M.P

E.D.D. CYCLES :

MARITAL HISTORY

OBSTETRIC INDEX

GRAVIDA: PARA: LIVE: ABORTION:

HISTORY:

PAST HISTORY : PIH / PRE ECLAMPSIA / RENAL DISEASE /

RECURRENT ABORTION, H/O MOTHER / SISTER / AUNT WITH H/O PRE ECLAMPSIA

FAMILY HISTORY : HYPERTENSION, EPILEPSY, DIABETES MELLITUS, RENAL DISEASE , OTHERS

MEDICAL HISTORY : HISTORY OF CHRONIC HYPERTENSION

DIABETES MELLITUS RENAL DISEASE HEART DISEASE

EPILEPSY

PERSONAL HISTORY: SMOKING/ ALCOHOL CONSUMPTION/ DRUG

OBSTETRIC HISTORY

PREVIOUS - PREGNANCY - DETAILED HISTORY

PRESENT OBSTETRIC HISTORY:

I TRIMESTER -FEVER, DRUG, INTAKE, RADIATION, BLEEDING PV

II TRIMESTER - BLEEDING, PEDAL EDEMA, VOMITTING, HEADACHE, VISUAL DISTURBANCES

III TRIMESTER - BLEEDING, PEDAL EDEMA, VOMITTING, HEADACHE, VISUAL DISTURBANCES

GENERAL EXAMINATION:

HEIGHT

WEIGHT

BUILT

NOURISHMENT

ANEMIA / JAUNDICE/LYMPHADENOPATHY

PEDAL EDEMA

THYROID

BREAST

C.V.S

R.S.

PULSE

BLOOD PRESSURE: LEFT LATERAL / SUPINE / SITTING PER ABDOMEN

TRIMESTER II/III

CORRESPONDING TO PERIOD TO AMMENORRHOEA

ABDOMINAL WALL EDEMA

FOETAL PRESENTATION

LIQOUR

FETAL HEART RATE

INVESTIGATION:

URINE - ALBUMIN

SUGAR DEPOSITS

HAEMOGLOBIN

COMPLETE BLOOD COUNT

BLOOD GROUPING AND Rh TYPING

SERUM CREATININE

BLOOD UREA

USG - PLACENTAL POSITION

FOLLOW UP AT TERM BLOOD PRESSURE

PROTEINURIA EDEMA

ABBREVIATIONS USED IN THIS DISSERTATION

ACOG	American College of Obstetrics and Gynaecology
BP	Blood Pressure
Fam.H/o PIH	Family History of PIH
hCG	Human Chorionic gonadotrophin
HT	Hypertension
PE	Pre-eclampsia
PIH	Pregnancy Induced Hypertension
Prev. PIH	Previous History of Pregnancy Induced Hypertension
Primi	Primigravida
PTH	Parathormone
p value	Probability value
WHO	World Health Organisation
H/o	History of
IL	Interleukin
TNF	Tumor Necrosis Factor
Vit. D	Vitamin D
CVS	Cardiovascular System
RS	Respiratory System
CNS	Central Nervous System
P/A	Per abdomen
US G	Ultrasonogram

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BIBLIOGRAPHY

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CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.N.Dhivya, PG in MD(OG)

Dear **Dr.N.Dhivya, PG in MD(OG)**

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"Lateral placelnta as a predictor of preeclampsia"

The following members of the ethics committee were present at the meeting held on 28.01.2008 at the Council Hall, Stanley Medical College, Chennai-1 at 10.00AM

Dr.C.B.Tharani, Director of Pharmacology,
Madras Medical College, Chennai-3 - Chairman of the Ethics Committee
Dr.S. Chitra, Vice-Principal,
Stanley Medical College, Chennai – 1- Member Secretary of the Ethics Committee

MEMBERS

Dr.Jayanthi
Prof.of Medical Gastroenterology
Dr.Madhavan
Prof.of Pharmacology
Dr.E.Dhandapani
Prof.of Medicine
Dr.Sujatha Sridharan
Prof.of Paediatrics
Thiru.Pachaiappan,
Junior Administrative Officer,
Thiru.A. Senthil Manoharan,
Advocate

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,

Member Secretary,
Ethics Committee

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

LOW RISK PREGNANCY GROUP

S.NO.	AGE	PARITY	2ND TRIMESTER		2ND VISIT BP	3RD VISIT BP	4TH VISIT BP	REMARKS
			BP	PLACENTA				
1.	21	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
2.	23	G2P1L1	110/70	CENTRAL	110/70	110/80	110/80	
3.	24	G3P1L1A1	120/80	CENTRAL	110/70	120/80	120/80	
4.	25	G2P1L1	120/80	**LATERAL**	100/60	120/80	**140/100**	**PRE ECLAMPSIA**
5.	23	PRIMI	120/70	CENTRAL	100/60	110/70	110/70	
6.	24	PRIMI	110/80	CENTRAL	110/70	120/80	120/80	
7.	16	PRIMI	120/80	CENTRAL	120/80	120/80	**140/90**	**PRE ECLAMPSIA**
8.	28	PRIM	120/80	CENTRAL	120/80	120/80	120/80	
9.	21	PRIMI	110/70	CENTRAL	120/70	110/80	110/80	
10.	39	PRIMI	120/80	CENTRAL	110/80	120/80	120/80	
11.	23	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
12.	24	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
13.	21	PRIMI	110/80	CENTRAL	110/70	120/80	120/80	
14.	23	PRIMI	120/80	CENTRAL	120/80	110/80	110/80	
15.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
16.	22	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
17.	32	PRIMI	120/80	CENTRAL	110/80	100/80	100/80	
18.	27	PRIMI	110/80	CENTRAL	120/80	120/70	120/70	
19.	22	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
20.	24	G3P1L1A1	120/80	CENTRAL	120/70	120/80	120/80	
21.	23	PRIMI	100/80	CENTRAL	120/80	120/80	120/80	
22.	16	PRIMI	120/70	CENTRAL	110/80	120/80	120/80	
23.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
24.	22	PRIMI	120/80	**LATERAL**	120/80	110/60	**140/100**	**PRE ECLAMPSIA**
25.	26	PRIMI	120/80	CENTRAL	100/80	120/80	120/80	
26.	34	G2P1L1	120/80	CENTRAL	120/70	120/80	120/80	
27.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
28.	23	PRIMI	110/60	CENTRAL	120/80	120/80	120/80	
29.	25	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
30.	21	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
31.	20	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
32.	24	PRIMI	120/80	CENTRAL	110/60	100/60	100/60	
33.	16	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
34.	23	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
35.	26	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
36.	24	PRIMI	100/60	CENTRAL	120/80	120/80	120/80	
37.	16	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
38.	24	PRIMI	120/80	**LATERAL**	120/80	110/80	**150/100**	**PRE ECLAMPSIA**
39.	27	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
40.	33	PRIMI	120/80	CENTRAL	100/60	120/80	120/80	
41.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
42.	21	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
43.	16	PRIMI	120/70	CENTRAL	120/80	110/80	110/80	
44.	25	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
45.	28	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
46.	23	PRIMI	120/80	CENTRAL	110/80	110/60	110/60	
47.	21	PRIMI	110/80	CENTRAL	120/70	120/80	120/80	
48.	24	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
49.	25	PRIMI	120/80	CENTRAL	120/80	100/70	100/60	
50.	32	G2P1L1	110/60	CENTRAL	120/80	110/70	100/70	
51.	22	G2P1L1	120/80	CENTRAL	110/80	110/80	120/80	
52.	23	G2P1L1	110/70	CENTRAL	120/70	120/70	110/80	
53.	17	PRIMI	120/80	CENTRAL	120/80	120/80	120/70	
54.	24	PRIMI	120/80	CENTRAL	110/60	110/60	**140/100**	**PRE ECLAMPSIA**
55.	29	G2P1L1	120/70	CENTRAL	120/80	120/80	110/60	
56.	17	PRIMI	110/80	CENTRAL	110/70	110/70	120/80	
57.	25	PRIMI	120/80	**LATERAL**	110/70	110/70	**140/90**	**PRE ECLAMPSIA**
58.	28	G3P1L1A1	120/80	CENTRAL	100/60	100/60	110/70	
59.	19	PRIMI	110/70	CENTRAL	100/60	100/60	100/60	
60.	24	PRIMI	120/80	CENTRAL	110/70	110/70	100/60	
61.	27	G2P1L1	120/80	CENTRAL	120/80	120/80	110/70	
62.	34	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
63.	23	PRIMI	110/80	CENTRAL	120/70	120/70	120/80	
64.	17	PRIMI	120/80	CENTRAL	110/80	110/80	120/70	
65.	23	G2P1L1	120/80	CENTRAL	120/80	120/80	110/80	

S.NO.	AGE	PARITY	2ND TRIMESTER		2ND VISIT BP	3RD VISIT BP	4TH VISIT BP	REMARKS
			BP	PLACENTA				
66.	26	PRIMI	120/70	**LATERAL**	120/80	120/80	**140/90**	**PRE ECLAMPSIA**
67.	24	PRIMI	120/80	CENTRAL	110/70	110/70	120/80	
68.	23	PRIMI	110/80	CENTRAL	120/80	120/80	110/70	
69.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
70.	22	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
71.	26	G2P1L1	100/80	CENTRAL	110/80	110/80	120/80	
72.	23	PRIMI	120/70	CENTRAL	120/80	120/80	110/80	
73.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
74.	27	G2P1L1	120/80	CENTRAL	120/70	120/70	120/80	
75.	18	PRIMI	120/80	CENTRAL	120/80	120/80	120/70	
76.	21	PRIMI	120/80	**LATERAL**	110/80	110/80	**150/100**	**PRE ECLAMPSIA**
77.	35	PRIMI	120/80	CENTRAL	120/80	120/80	110/80	
78.	16	PRIMI	110/60	CENTRAL	120/80	120/80	120/80	
79.	20	PRIMI	120/80	CENTRAL	100/80	100/80	120/80	
80.	29	G3P2L2	120/80	CENTRAL	120/70	120/70	100/80	
81.	18	PRIMI	120/80	CENTRAL	120/80	120/80	**150/100**	**PRE ECLAMPSIA**
82.	21	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
83.	28	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
84.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
85.	30	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
86.	18	PRIMI	100/60	CENTRAL	110/60	110/60	120/80	
87.	22	G3P2L2	120/80	CENTRAL	120/80	120/80	110/60	
88.	21	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
89.	30	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
90.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
91.	23	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
92.	24	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
93.	34	PRIMI	120/70	**LATERAL**	120/80	**150/90**	**170/100**	**SEVERE**
94.	24	PRIMI	120/80	CENTRAL	100/60	100/60	120/80	**PREECLAMPSIA**
95.	30	G2P1L0	120/80	CENTRAL	120/80	120/80	100/60	
96.	17	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
97.	24	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
98.	29	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
99.	25	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
100.	18	PRIMI	110/60	CENTRAL	110/80	110/80	120/80	
101.	18	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
102.	25	G2P1L0	110/70	CENTRAL	120/70	110/80	110/80	
103.	29	PRIMI	120/80	CENTRAL	110/80	120/80	120/80	
104.	30	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
105.	23	PRIMI	120/70	CENTRAL	120/80	110/70	110/70	
106.	17	PRIMI	110/80	CENTRAL	110/70	120/80	120/80	
107.	24	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
108.	29	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
109.	16	PRIMI	110/70	CENTRAL	120/80	110/80	110/80	
110.	23	G2P1L1	120/80	CENTRAL	110/80	120/80	120/80	
111.	28	PRIMI	120/80	**LATERAL**	120/80	120/80	**150/100**	**PRE ECLAMPSIA**
112.	18	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
113.	22	G3P2L1A1	110/80	CENTRAL	120/70	120/80	120/80	
114.	27	G2P1L1	120/80	CENTRAL	120/80	110/80	110/80	
115.	24	PRIMI	120/80	CENTRAL	110/80	120/80	120/80	
116.	23	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
117.	33	PRIMI	120/80	CENTRAL	120/80	100/80	100/80	
118.	24	G2P1L1	110/80	CENTRAL	100/80	120/70	120/70	
119.	23	G2P1L1	120/80	CENTRAL	120/70	120/80	**150/100**	**PRE ECLAMPSIA**
120.	24	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
121.	18	PRIMI	100/80	CENTRAL	120/80	120/80	120/80	
122.	21	PRIMI	120/70	**LATERAL**	120/80	120/80	**150/100**	**PRE ECLAMPSIA**
123.	26	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
124.	23	G3P1L1A1	120/80	CENTRAL	120/80	110/60	110/60	
125.	24	G2P1L1	120/80	CENTRAL	110/60	120/80	120/80	
126.	17	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
127.	20	PRIMI	120/80	CENTRAL	120/80	120/80	**140/90**	**PRE ECLAMPSIA**
128.	28	PRIMI	110/60	CENTRAL	120/80	120/80	120/80	
129.	23	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
130.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
131.	21	PRIMI	120/80	**LATERAL**	**140/90**	**140/90**	**150/100**	**ONSET AT 28**
132.	27	G2P1L1	120/80	CENTRAL	120/80	100/60	100/60	**WEEKS**
133.	24	PRIMI	120/80	CENTRAL	100/60	120/80	120/80	
134.	18	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	

S.NO.	AGE	PARITY	2ND TRIMESTER		2ND VISIT BP	3RD VISIT BP	4TH VISIT BP	REMARKS
			BP	PLACENTA				
135.	22	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
136.	28	G2P1L1	100/60	CENTRAL	120/80	120/80	120/80	
137.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
138.	18	PRIMI	120/80	CENTRAL	120/80	110/80	110/80	
139.	23	G2P1L0	120/80	CENTRAL	110/80	120/70	120/70	
140.	29	G2P1L1	120/80	CENTRAL	120/70	120/80	120/80	
141.	20	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
142.	21	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
143.	18	PRIMI	120/70	CENTRAL	120/80	110/80	110/80	
144.	24	PRIMI	120/80	CENTRAL	110/80	120/70	120/70	
145.	30	G2P1L1	120/80	CENTRAL	120/70	120/80	120/80	
146.	24	G2P1L1	120/80	CENTRAL	120/80	110/60	110/60	
147.	18	PRIMI	110/80	CENTRAL	110/60	120/80	120/80	
148.	25	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
149.	29	G2P1L0	120/80	CENTRAL	110/70	100/70	100/70	
150.	21	G2P1L1	110/60	CENTRAL	110/70	110/70	110/70	
151.	18	PRIMI	120/80	CENTRAL	100/60	110/80	110/80	
152.	23	PRIMI	110/70	CENTRAL	100/60	120/70	120/70	
153.	28	PRIMI	120/80	**LATERAL**	110/70	**140/90**	**150/100**	**PRE ECLAMPSIA**
154.	19	PRIMI	120/80	CENTRAL	120/80	110/60	110/60	
155.	24	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
156.	19	G2P1L1	110/80	CENTRAL	120/70	110/70	110/70	
157.	25	PRIMI	120/80	CENTRAL	110/80	110/70	110/70	
158.	27	PRIMI	120/80	CENTRAL	120/80	100/60	100/60	
159.	19	PRIMI	110/70	CENTRAL	120/80	100/60	100/60	
160.	24	PRIMI	120/80	CENTRAL	110/70	110/70	110/70	
161.	34	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
162.	26	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
163.	26	PRIMI	110/80	CENTRAL	120/80	120/70	120/70	
164.	17	PRIMI	120/80	CENTRAL	110/80	110/80	110/80	
165.	23	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
166.	27	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
167.	24	PRIMI	120/80	CENTRAL	120/70	110/70	**140/90**	**PRE ECLAMPSIA**
168.	19	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
169.	22	G2P1L1	120/80	CENTRAL	110/80	120/80	120/80	
170.	28	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
171.	17	PRIMI	100/80	CENTRAL	120/80	110/80	110/80	
172.	21	PRIMI	120/70	CENTRAL	100/80	120/80	120/80	
173.	29	PRIMI	120/80	CENTRAL	120/70	120/80	120/80	
174.	35	G2P1L1	120/80	CENTRAL	120/80	120/70	120/70	
175.	21	PRIMI	120/80	CENTRAL	120/80	120/80	**150/100**	**PRE ECLAMPSIA**
176.	22	PRIMI	120/80	CENTRAL	120/80	110/80	110/80	
177.	17	PRIMI	120/80	CENTRAL	120/80	120/80	**150/90**	**PRE ECLAMPSIA**
178.	20	PRIMI	110/60	CENTRAL	120/80	120/80	120/80	
179.	30	G2P1L1	120/80	CENTRAL	110/60	100/80	100/80	
180.	24	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
181.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
182.	21	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
183.	29	G3P1L1A1	120/80	CENTRAL	120/80	120/80	120/80	
184.	36	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
185.	24	G2P1L1	120/80	CENTRAL	120/80	120/80	**150/100**	**PRE ECLAMPSIA**
186.	23	PRIMI	100/60	CENTRAL	120/80	110/60	110/60	
187.	18	PRIMI	120/80	CENTRAL	100/60	120/80	120/80	
188.	22	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
189.	34	PRIMI	120/80	**LATERAL**	120/80	120/80	**160/110**	**ECLAMPSIA**
190.	28	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
191.	18	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
192.	23	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
193.	27	G2P1L1	120/70	CENTRAL	110/80	120/80	150/90	
194.	28	PRIMI	120/80	CENTRAL	120/80	100/60	100/60	
195.	18	PRIMI	120/80	CENTRAL	120/70	120/80	120/80	
196.	24	G3P2L2	120/80	CENTRAL	110/80	120/80	120/80	
197.	18	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
198.	25	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
199.	31	PRIMI	120/80	CENTRAL	110/70	120/80	120/80	
200.	18	PRIMI	110/60	CENTRAL	120/80	110/80	110/80	
201.	31	G2P1L1	120/80	CENTRAL	120/80	120/70	120/70	
202.	23	PRIMI	110/70	CENTRAL	120/80	110/80	110/80	
203.	24	PRIMI	120/80	CENTRAL	110/80	120/80	120/80	

S.NO.	AGE	PARITY	2ND TRIMESTER		2ND VISIT BP	3RD VISIT BP	4TH VISIT BP	REMARKS
			BP	PLACENTA				
204.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
205.	23	PRIMI	120/70	**LATERAL**	120/80	110/70	**140/90**	**PRE ECLAMPSIA**
206.	26	G2P1L1	110/80	CENTRAL	120/70	120/80	120/80	
207.	18	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
208.	22	PRIMI	120/80	CENTRAL	110/80	120/80	120/80	
209.	27	PRIMI	110/70	CENTRAL	120/80	110/80	110/80	
210.	21	G2P1L1	120/80	CENTRAL	120/80	120/80	**140/100**	**PRE ECLAMPSIA**
211.	22	PRIMI	120/80	CENTRAL	100/80	120/80	**140/100**	**PRE ECLAMPSIA**
212.	17	PRIMI	120/80	CENTRAL	120/70	120/70	120/70	
213.	21	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
214.	28	G2P1L1	120/80	CENTRAL	120/80	110/80	110/80	
215.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
216.	32	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
217.	21	PRIMI	120/80	CENTRAL	120/80	100/80	100/80	
218.	21	PRIMI	110/80	CENTRAL	110/60	120/70	120/70	
219.	16	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
220.	20	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
221.	29	PRIMI	100/80	**LATERAL**	120/80	**140/90**	**140/90**	**PRE ECLAMPSIA**
222.	23	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
223.	23	G3P1L1A1	120/80	CENTRAL	120/80	120/80	**140/100**	**PRE ECLAMPSIA**
224.	21	PRIMI	120/80	CENTRAL	120/80	110/60	110/60	
225.	18	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
226.	21	G2P1L1	120/80	CENTRAL	100/60	120/80	120/80	
227.	30	PRIMI	120/80	**LATERAL**	120/80	120/80	**140/100**	**PRE ECLAMPSIA**
228.	33	PRIMI	110/60	CENTRAL	120/80	120/80	120/80	
229.	29	G3P2L2	120/80	CENTRAL	120/80	120/80	120/80	
230.	22	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
231.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	

HIGH RISK PREGNANCY GROUP

S.N0	AGE	PARITY	2ND TRIMESTER		2ND VISIT BP	3RD VISIT BP	4TH VISIT BP	HIGH RISK
			BP	PLACENTA				
1.	18	PRIMI	120/80	CENTRAL	120/80	120/70	120/80	GDM
2.	28	G2P1L1	110/70	CENTRAL	110/70	110/80	120/80	
3.	23	PRIMI	120/80	LATERAL	110/70	120/80	**150/100**	
4.	24	G3A2	120/80	LATERAL	100/60	120/80	**150/90**	BOH
5.	34	PRIMI	120/70	CENTRAL	100/60	110/70	120/80	GDM
6.	23	PRIMI	110/80	CENTRAL	110/70	120/80	**140/90**	
7.	21	PRIMI	120/80	CENTRAL	120/80	120/80	120/70	
8.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
9.	21	PRIMI	110/70	CENTRAL	120/70	110/80	110/80	
10.	18	PRIMI	120/80	CENTRAL	110/80	120/80	120/80	
11.	29	G4P1L1A2	120/80	CENTRAL	120/80	120/80	120/80	BOH
12.	23	PRIMI	120/80	CENTRAL	120/80	120/70	100/80	GDM
13.	24	PRIMI	110/80	LATERAL	110/70	120/80	**140/90**	GDM
14.	36	PRIMI	120/80	CENTRAL	120/80	110/80	**150/100**	GDM
15.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
16.	18	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	
17.	30	G2P1L1	120/80	CENTRAL	110/80	100/80	120/80	
18.	24	PRIMI	110/80	CENTRAL	120/80	120/70	120/80	GDM
19.	23	PRIMI	120/80	CENTRAL	120/80	120/80	110/60	
20.	29	PRIMI	120/80	CENTRAL	120/70	120/80	120/80	
21.	19	PRIMI	100/80	CENTRAL	120/80	120/80	120/80	
22.	28	G3A2	120/70	CENTRAL	110/80	120/80	120/80	BOH
23.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
24.	23	G3P1L1A1	120/80	LATERAL	120/80	110/60	**140/100**	GDM
25.	19	PRIMI	120/80	CENTRAL	100/80	120/80	120/80	
26.	27	PRIMI	120/80	CENTRAL	120/70	120/80	120/80	INFERTILITY
27.	23	PRIMI	120/80	CENTRAL	120/80	120/80	100/60	
28.	40	PRIMI	110/60	CENTRAL	120/80	120/80	120/80	
29.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	**BOH**
30.	26	G4A3	120/80	CENTRAL	120/80	120/80	120/80	GDM
31.	23	PRIMI	120/80	LATERAL	120/80	120/80	**150/100**	
32.	19	PRIMI	120/80	CENTRAL	110/60	100/60	120/80	
33.	19	PRIMI	120/80	CENTRAL	120/80	120/80	110/80	
34.	27	G3A2	120/80	CENTRAL	120/80	120/80	120/70	**BOH**
35.	28	G4A3	120/80	CENTRAL	120/80	120/80	120/80	**BOH**
36.	24	G4P1LOA2	100/60	CENTRAL	120/80	120/80	120/80	**BOH**
37.	19	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
38.	29	PRIMI	120/80	CENTRAL	120/80	110/80	**150/90**	GDM
39.	19	PRIMI	120/80	CENTRAL	120/80	120/70	120/70	
40.	30	G3P1L0A1	120/80	CENTRAL	100/60	120/80	120/80	**BOH**
41.	23	PRIMI	120/80	CENTRAL	120/80	120/80	**150/90**	
42.	38	PRIMI	110/80	LATERAL	120/80	120/80	**170/110**	**INFERTILITY**
43.	24	G4P1LOA2	120/70	CENTRAL	120/80	110/80	110/70	GDM,BOH
44.	18	PRIMI	120/80	CENTRAL	120/80	120/70	120/80	
45.	29	G2P1L1	120/80	CENTRAL	120/80	120/80	120/80	
46.	23	PRIMI	120/80	CENTRAL	110/80	110/60	**140/100**	
47.	16	PRIMI	110/80	CENTRAL	120/70	120/80	110/80	
48.	28	G4A3	120/70	CENTRAL	120/80	120/80	120/80	BOH
49.	23	PRIMI	120/80	LATERAL	120/80	100/70	**140/90**	
50.	24	G3A2	110/60	LATERAL	120/80	110/70	**140/90**	**BOH**
51.	18	PRIMI	120/80	CENTRAL	110/80	110/80	120/80	
52.	26	G3P1LOA1	110/70	CENTRAL	120/70	120/70	120/80	GDM,BOH
53.	23	PRIMI	120/80	CENTRAL	120/80	120/80	120/80	
54.	24	PRIMI	120/80	LATERAL	110/60	110/60	**140/100**	GDM &
55.	32	PRIMI	120/70	CENTRAL	120/80	120/80	120/80	INFERTILITY
56.	24	PRIMI	110/80	CENTRAL	110/70	110/70	120/80	
57.	23	PRIMI	120/80	CENTRAL	110/70	110/70	120/70	

S.N0	AGE	PARITY	2ND TRIMESTER		2ND VISIT BP	3RD VISIT BP	4TH VISIT BP	HIGH RISK
			BP	PLACENTA				
58.	24	PRIMI	120/80	LATERAL	100/60	100/60	**150/100**	GDM
59.	24	PRIMI	110/70	CENTRAL	100/60	100/60	110/80	
60.	27	PRIMI	120/80	CENTRAL	110/70	110/70	120/80	BOH
61.	17	G3A2	120/80	CENTRAL	120/80	120/80	120/80	
62.	23	PRIMI	120/80	CENTRAL	120/80	120/80	110/70	
63.	22	PRIMI	110/80	CENTRAL	120/70	120/70	120/80	
64.	22	PRIMI	120/80	CENTRAL	110/80	110/80	120/80	
65.	24	PRIMI	120/80	CENTRAL	120/80	120/80	120/70	
66.	21	PRIMI	120/70	CENTRAL	120/80	120/80	110/80	GDM
67.	22	PRIMI	120/80	CENTRAL	110/70	110/70	120/80	
68.	24	PRIMI	110/80	CENTRAL	120/80	120/80	120/80	
69.	24	PRIMI	120/80	CENTRAL	120/80	120/80	110/70	